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Disease-a-Month

Thermal Burns

STANLEY M. LEVENSON
CHARLES C. LUND

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Thermal Burns

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IN 1956, as in most years of the preceding decade, some 7,000 people died of burns in the United States; the total number of seriously burned patients is probably about 150,000 yearly. It has been estimated that 6,000-16,000 hospital beds are occupied each day through the year by the burned and scalded (1), but figures alone do not convey the gravity of the situation, nor the suffering, the disfigurement and the economic losses.

About two thirds of all fatal burns result from home accidents; burns are the second most common type of home injury, and they happen most frequently to young children and to aged persons because of their relative helplessness. One quarter of those who die from burns received in the home are children up to 4 years of age and one quarter are people over 65. The responsibility for these injuries clearly falls on those charged with the daily care of these people. An intensive educational program directed toward the prevention of burns must be vigorously carried on; it is only by successful preventive

measures that we can hope to substantially reduce the injuries and deaths from burns in the near future.

It appalls one to contemplate the number of people who might be burned in a future war in which nuclear weapons are used. Burns, it has been estimated, were responsible for more than half the fatalities and about three quarters of all the casualties at Hiroshima and Nagasaki. This means that about 150,000 people were burned, 50,000 of whom died. Some had, as well, other types of injuries and radiation damage.

In this discussion, attention will be directed almost entirely to the problems of thermal burns encountered in ordinary civilian life; because of limitations of space, only brief mention will be made of burns resulting from nuclear explosions despite their obvious importance.

LOCAL PATHOLOGY

The extent of injury to a burned tissue depends on the intensity, quality and duration of the thermal stimulus. There is a gradation of effects, from the simple reversible dilatation and superficial cellular damage to the marked increase of capillary permeability, thromboses and extensive cellular destruction characteristic of deep burns. Burns are classified into the 3 following categories, according to their depth: first degree, simple erythema; second degree, partial destruction of the skin, but without destruction of all the deep epithelial cells; third degree, destruction of the full thickness of the skin.

Evans and his associates (2) have measured the incident thermal dose (calories per unit area) required to produce first, second and third degree flash burns on the unprotected skin of human volunteers. They found that thermal doses in excess of 2.0 cal. per square centimeter delivered in 0.5 second were required to produce a first degree burn; 3.2 cal., superficial second degree; 3.9 cal., well-defined second degree; 4.8 cal., deep second degree and possibly third degree. When these same doses were delivered over a longer time, the severity of the burns decreased.

The immediate response to the mildest burn is a brief period of vasoconstriction; dilatation of the capillaries, the finer arterioles and venules follows, with markedly increased blood flow. If the injury is somewhat more severe, capillary permeability is locally increased, and plasma-like fluid leaks into, or out of, the tissue. (Its electrolyte pattern is similar to plasma, but its

protein content is lower, but with a higher proportion of albumin.) At the same time, there is an abnormal exchange of certain constituents between the extracellular and intracellular fluids (e.g., sodium, potassium and certain proteolytic enzymes). Present evidence indicates no generalized loss of fluid in unburned regions. A certain number of red cells in the capillaries may be damaged; sludging and thrombosis may occur.

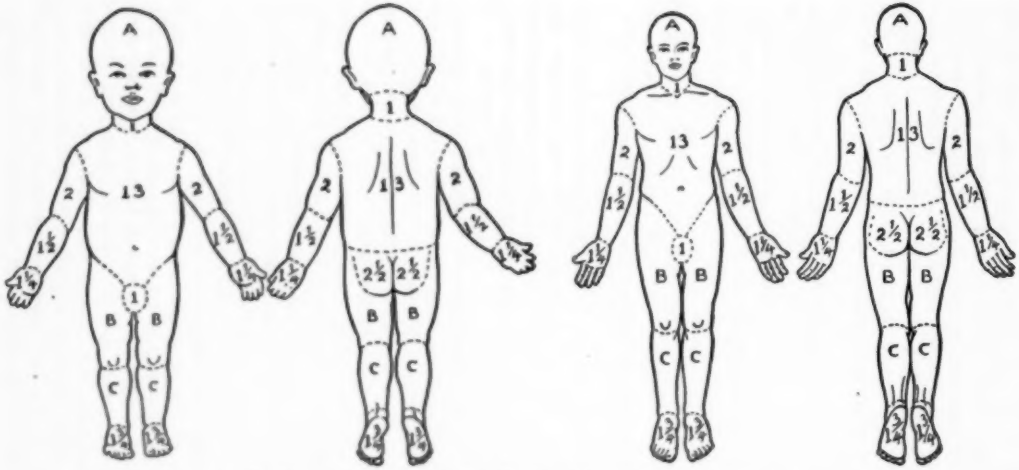
The tissue space becomes distended with fluid from leaking capillaries. If this fluid is near the surface, it forms blisters which finally rupture. Capillary leakage at deeper levels results in local edema; some fluid finds its way into the lymphatic system, giving rise to greatly increased lymph flow from the burned area (3, 4). The tissue cells and intercellular substances, such as collagen, are denatured and injured to varying degrees depending on the intensity and duration of burn. A full-blown inflammatory reaction with the usual leukocytic response sets in.

Healing of first degree burns proceeds rapidly and is complete in a few days; there is a return of normal tone to the blood vessels, cessation of the very slight leakage, absorption of any excess intercellular fluid and desquamation of the very superficially injured epithelium.

In superficial second degree burns, although the necrosis extends through the epidermis into the derma, there are many viable epithelial remnants. Hyperplasia of epithelial cells may occur within 24 hours, and epithelization takes place within 2 weeks; such burns heal without scarring.

In deep second degree burns, on the other hand, there are relatively few epithelial remnants. Often, only the deepest parts of the sweat glands and hair follicles remain viable. In addition, the necrosed superficial layer must separate before epithelization can occur. Consequently, healing is delayed, and there may be considerable fibrous tissue formation leading to scarring and impaired function.

In third degree burns, the entire thickness of skin and all its epithelial elements are destroyed. The slough is thicker than in deep second degree burns and its separation proceeds more slowly. The dead cells are removed by lysis and phagocytosis; the intercellular collagen resists digestion more than the cells and the collagen fibers keep the slough attached until they are finally loosened at the junction of living and dead tissue. After the slough has separated, spontaneous epitheliza-



RELATIVE PERCENTAGES OF AREAS AFFECTED BY GROWTH

AREA	AGE		
	0	1	5
A = ½ of Head	9½	8½	6½
B = ½ of One Thigh	2¾	3¼	4
C = ½ of One Leg	2½	2½	2¾

AREA	AGE		
	10	15	Adult
A = ½ of Head	5½	4½	3½
B = ½ of One Thigh	4¼	4½	4¾
C = ½ of One Leg	3	3¼	3½

FIG. 1.—Diagrams for charting burned areas.

tion of the denuded surface can take place only from the edges of the wound; therefore, deep burns of any significant size must be grafted.

It is frequently impossible to judge correctly the depth of the burn at the time of injury. The typical dermal burn is characterized by the presence of many small broken or intact blebs, visible pink corium and slight subcutaneous edema. In contrast, the typical deep flame burn is characterized by a dry, dead white, brown or charred appearance with beginning

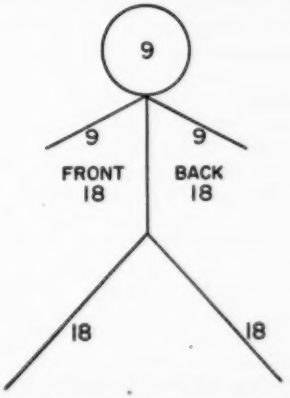


FIG. 2.—Rule of 9's for estimating area of skin surface burned.

subcutaneous edema. But many burns, particularly the deep dermal variety, do not show these typical appearances; these cannot be definitely classified for several days.

Bull and Lennard-Jones (5) have described a method for estimating the depth of skin burn based on the observation that burns of different depths retain different pain sensitivities to pin prick. There is usually moderate hypalgesia in partial thickness burns and marked hypalgesia or anesthesia in full thickness burns. These differences are observed chiefly during the period 2 hours and 2 days after burning, but there is great variation in response within the first 1 or 2 hours. After 2 days, pain sensation may be normal in partial thickness burns, but the diminished pain sensation in full thickness burns continues until much later. This technic has the limitations of any test which depends on subjective responses of the patient.

Hemoglobinemia and hemoglobinuria usually denote a deep, extensive burn, but their occurrence gives no information as to the depth in any particular area.

The severity of a burn depends not only on its depth but also on its extent. To provide guides for estimating the area

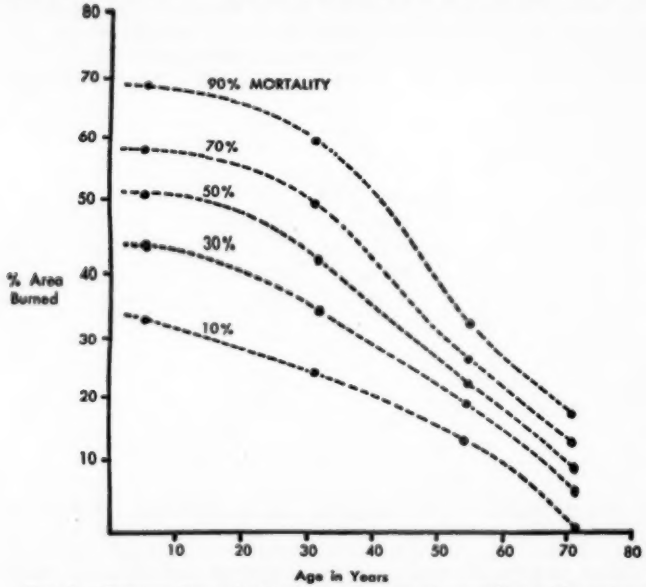


FIG. 3.—Contour lines dividing zones of equal mortality with various combinations of age and severity. (Reprinted from Bull, J. P., and Squire, J. R.: A study of mortality in a burns unit; standards for the evaluation of alternative methods of treatment, Ann. Surg. 130:160, 1949.)

of skin surface burned, Weidenfield in 1902 and Berkow in 1928 measured the total surface areas of many people, and the areas of discrete parts of their bodies. Diagrams for charting burned areas based on these and related information are here reproduced from an article by Lund and Browder (6) (Fig. 1). The "Rule of 9's" has been recently introduced to make possible an even quicker estimation in event of mass disaster (Fig. 2). This is roughly correct for adults and older children, but is a poor approximation for infants.

There may be considerable variations in response to burns of apparently equal depth and extent among different patients, depending, in part, on age and prior state of health. Mortality is disproportionately high among the elderly (Fig. 3) and the infirm.

RESPIRATORY TRACT INJURY

Respiratory tract injury is a serious occurrence in some burned patients. The Cocoanut Grove fire of 1942 in Boston, Massachusetts provided the largest single experience with this injury in recent years. Of 131 burned patients admitted to the Boston City Hospital, 107 had some degree of respiratory involvement; in 55, the respiratory involvement was severe (7). A similar distribution occurred in the patients admitted at the same time to the Massachusetts General Hospital (8). Among these patients, the fundamental pathologic lesion was a necrotizing laryngotracheobronchitis, with the formation of a pseudodiphtheritic membrane; many of the respiratory passages were occluded by the pseudomembrane and viscid, sanguineous, mucoid exudate. There were scattered areas of congestion, atelectasis, emphysema and hemorrhage. The usual symptoms were sore throat, cough, hoarseness, dyspnea, cyanosis, wheezing, stridor, raising of sputum (mucoid, purulent, bloody) and restlessness. Scattered areas of dullness, hyperresonance and rales of all varieties were commonly found. Scattered areas of atelectasis, emphysema, congestion and consolidation were observed by x-ray. Secondary pulmonary infections were few and usually mild, presumably due to the early and sustained chemotherapy with sulfonamides. The respiratory damage was probably due to irritants in the fumes and particles from the fire. From experimental studies by Moritz and associates (9) it is unlikely that inhalation of hot air, as such, was an important factor. The victims who lost consciousness sustained the severest respiratory damage; presumably they were exposed longest and thus inhaled the most fumes. Similar pathologic findings were reported from the Cleveland Clinic fire of 1928. This type of injury is not caused exclusively, however, by mass conflagrations; it may be a complication of burns quite "ordinarily" come by. Injury of the respiratory tract should be suspected in any patient who has inhaled large amounts of hot smoke and particularly if he has lost consciousness. This complication arises most often

in patients with face burns, but at times it occurs in patients with no external burns at all. Many of them have singed nasal hair, soot on their faces and burns of the nasopharynx.

If injury of the respiratory tract of a patient is suspected, he must be watched closely. All personnel who come in contact with him should be masked. A tracheotomy kit should be kept at his bedside. His nose, nasopharynx, epiglottis, larynx and vocal cords should be examined. If his nasopharynx is burned, his nose should be swabbed with bland oil; it sometimes helps to have him gargle with a warm, soothing solution such as glucose in water.

Respirations should be checked every half hour. The air in the room should be humidified. The patient should be turned frequently and encouraged to cough. If any hoarseness, dyspnea or stridor occurs, he should be placed in an oxygen tent. Helium-oxygen mixtures may be more easily breathed than 100% oxygen. If there is evidence of bronchial spasm, intravenous aminophylline should be tried, or adrenalin or ephedrine by nebulizer. Deep respirations should be induced for a few minutes every 1-2 hours by having the patient inhale a 10% carbon dioxide-90% oxygen mixture. Oxygen under pressure, together with a detergent and antispasmodic agent, may be useful. Generalized laryngotracheobronchial spasm may seize the patient suddenly and may kill him unless it is relieved immediately by tracheotomy and/or artificial respiration. Tracheotomy is necessary if stridor, air hunger or other signs of laryngeal obstruction appear. One should do a tracheotomy early if indicated; if the question arises whether a tracheotomy should be performed, it should be done at once. Suction through the tracheotomy tube should be done repeatedly as needed; when simple aspiration fails, bronchoscopic aspirations may succeed.

Restlessness of these patients may be due primarily to lack of oxygen, in which case oxygen supply should be increased as outlined above; or to pain (from surface burns), for which Demerol®, rather than morphine, should be used; or to fear and nervousness, in which case barbiturates should be given.

Parenteral penicillin, in large dosages, should be started on admission. Antibiotic therapy might be altered later, if indicated by bacteriologic studies.

If the patient with injury of the respiratory tract also has extensive surface burns, appropriate fluid (colloid and electrolyte) should be given without hesitation, but cautiously,

to prevent or treat shock. However, the patient must be watched closely for pulmonary edema.

Occurrence of residual damage from the respiratory tract injury is apparently neither frequent nor severe. Information on this point was gathered over a 2-year period following the Cocoanut Grove fire. While most of the patients raised little or no sputum with the cough, particularly during the first few months, an occasional patient later began to cough copious amounts of mucopurulent sputum, suggesting the development of bronchiectasis. Residual chronic nasopharyngitis, and possibly the development of mild chronic sinusitis, was suggested by postnasal discharge in a few of the patients. Follow-up roentgen examination in inspiration and expiration revealed no residual changes.

HEMOGLOBINEMIA, HEMOGLOBINURIA AND EARLY ANEMIA

Hemoglobinemia and hemoglobinuria were found in severely burned patients over a century ago. In general, the former is seen in patients with deep burns involving 10% or more of the body area, and the latter in patients with such burns involving 30% area or more.

Lyon and his associates (10) have recently worked out a quantitative relation of the degree of hemoglobinemia to the volume of injured tissue in burned swine.

Hemoglobinemia and hemoglobinuria are maximal soon after the burn and then decrease gradually during the first 24-72 hours. The red cell pigments in the plasma and the urine are oxyhemoglobin mixed with traces of methemoglobin.

The possible relationship of hemoglobinemia and hemoglobinuria to kidney function will be discussed later.

The blood of severely burned patients may show early alterations that vary from only slight spherocytosis of erythrocytes to advanced spherocytosis and fragmentation. There may be a marked increase in osmotic fragility, involving as much as 30% of the patient's red cells. Correspondingly, blood destruction may be of a mild degree with little hemoglobinemia and no hemoglobinuria, or a considerable mass of red cells may be quickly destroyed, followed by marked hemoglobinemia and hemoglobinuria.

Studies of the mechanism of these changes in the red blood cells have revealed no cold or warm agglutinins or hemolysins.

Shen and associates (11) have concluded that the destruction of a considerable volume of erythrocytes may result from heating of blood at the site of the burn, depending on at least 3 major factors: the temperature attained by the blood, the duration of heating and the volume of blood subjected to these conditions. They found that if blood is rapidly heated to a temperature of 51-65 C., red cells fragment, form spherocytes and microspherocytes, become fragile and hemolyze. The red cells of patients with hemoglobinemia, examined promptly after the burn, exhibited these same changes.

Studies of erythrocyte survival by tracer experiments have shown that about 10-20% of the circulating erythrocytes may be destroyed in the early days after burning (12). The evidence regarding erythropoiesis during this early period is controversial (13). This early anemia is often greater in patients treated for shock with plasma than in patients treated with type-specific blood; this is probably related to the fact that pooled plasma given to injured patients with types A, B or A-B blood may induce a hemolytic process.

BURN SHOCK

Most patients who die within the first 48 hours after being burned, die of shock. The important initiating factors are loss of fluid, sodium and protein from the vascular bed, with resulting oligemia, hyponatremia and hypoproteinemia. Under certain conditions, other elements may be important.

The amount of fluid leaking into, or from, a burned area depends on the extent of the area, the vascularity of the tissue and the depth of the burn. Superficial second degree burns are characterized by external fluid loss, while subcutaneous seepage results from deeper burns. There is no evidence that the adrenal cortical hormones influence permeability of the damaged capillary or the resultant fluid loss.

The maximum loss occurs in the first 12 hours; the rate then slows down. Millican (14) has shown that the burn edema fluid sodium is rapidly exchangeable with that of the plasma. (Presumably, this is true of other electrolytes and water.) The protein is only slowly exchangeable. Resorption of the edema fluid begins about 36-48 hours after injury. It proceeds slowly, and usually takes 5 to 7 days. Figure 4 presents data on a 55-year-old male with second degree burns of face and hands (7% body area) illustrating some of these points.

The amount of fluid loss and reduction in blood volume is usually indicated by a rising hematocrit, hemoglobin and red blood cell count. However, the apparent hemoconcentration may not parallel the oligemia, because measurements of

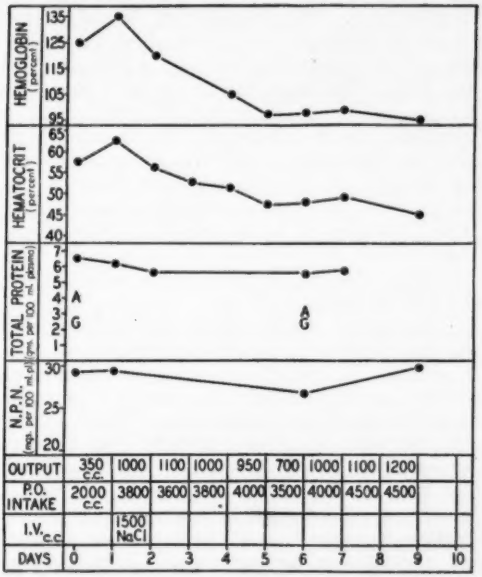


FIG. 4.—Hematologic changes in patient with burns of face and hands and without shock.

hematocrit and hemoglobin in samples obtained from peripheral vessels may not reflect their true "body" values adequately. If extensive red cell lysis has occurred, hematocrit determinations will be misleading. Also, hemoconcentration may not be apparent despite severe losses of plasma in patients anemic prior to burning. A rapid exchange of fluid may be taking place without much change in hematocrit; return of fluid to the vascular bed may keep pace with the loss. This will be indicated by a progressive fall in plasma protein concentration, since the concentration of protein in the returning extravascular fluid is lower than in plasma.

The literature concerning toxic factors in burn shock is voluminous and controversial. Christophe and Prinzmetal have reported experiments which appear to support the possibility of toxins. Christophe (15) has described the production of death from "burn shock" in dogs perfused with blood from severely burned dogs; Prinzmetal (16) found that rats with a single severely burned hind limb died in shock without visible edema and apparently without sufficient fluid loss to account for death. The term "toxin" is used in a broad sense to signify any agent or agents which adversely affect important bodily functions. Thus, substances which are "nontoxic" in their normal environment or usual concentrations may become toxic when present in unusual concentrations or in unusual environments. There is little doubt that many chemicals are released from the area of burn—but their physiologic significance is not known. Among these substances are various proteolytic enzymes, lipases, alkaline phosphatase, nucleic acid metabolites and histamine (17, 18).

A large number of studies have appeared which compare the physiologic effects of extracts of normal and burned skin of various species. The results are controversial and confusing. One important variable which has not been controlled adequately is infection of the burned skin. There is little doubt that certain bacteria, in large numbers, will produce toxins which may result in shock; it has not been possible to differentiate this type of toxin from that arising from the burned area independent of infection, since all deep burns are infected. Studies on germ-free animals will be illuminating in this regard and also in further investigations of the concept of Fine (19) that "irreversibility" of hemorrhagic shock (and presumably of other forms of shock) is due to the entrance into the circulation of certain bacterial endotoxins.

Neurogenic influences appear to be relatively unimportant in the production of burn shock.

A burned patient in fully developed shock is apathetic, chilly, often nauseated, pale and cyanotic. His blood pressure is low, his pulse rapid and thready. Venous pressure is high, cardiac output low, peripheral resistance high, peripheral blood flow decreased and urinary output low.

The diagnosis of burn shock at this stage is easy, but to wait for this stage to appear is analogous to waiting for the appearance of a cavity in the lung before making the diagnosis of pulmonary tuberculosis. If shock persists, irreversible changes

in the function and structure of vital organs may result, after which remedial measures become impossible. The problem must be attacked by preventing shock.

Shock may be anticipated in previously normal adults with a burned area of 15% or greater. In the elderly or the very young, a burned area of as little as 8% may lead to circulatory failure if untreated. Any procedure that may increase the shock (e.g., dressings) should be avoided or postponed until shock has been prevented or treated. Restoration and maintenance of blood volume and correction of electrolyte and colloid disturbances are positive measures of primary importance.

In the period 1925-1940, intravenous saline had been used as the primary replacement fluid in the therapy of burn shock to counteract hyponatremia, hypochloremia and oligemia. To a lesser extent, whole blood was also used, in part because of the expected anemia.

When plasma became generally available in the 1940's, it at first completely replaced these solutions. Since the fluid lost from the vascular bed is similar in composition to blood plasma, it seemed logical to replace it with plasma. It also appeared to some that when intravenous saline was used, the therapeutic effect was frequently transient, and that plasma protein was "washed out." In regard to the use of whole blood, it had been felt by some clinicians that since hemoconcentration was already present in the severely burned patient, additional red blood cells would serve no useful purpose. However, in recent years, the dogmatism of the 1940's has been questioned, and there has been considerable discussion about the relative roles of the plasma protein (or colloid) component of the plasma and its sodium content and of red blood cells.

The work of Rosenthal (20) revived interest in the use of sodium chloride solution alone or in conjunction with other sodium salts. He showed that the therapeutic effects of sodium salts given to rodents and man intraperitoneally, orally or intravenously after burns are similar to those of serum or plasma. In some experiments, the effect of serum appeared to be due not to its protein but to the sodium, while in other experiments, when the fluids were administered to mice in divided doses, both serum and dextran gave better therapeutic responses than equivalent amounts of saline, when less than full therapeutic amounts of either were given. The therapeutic effect of the sodium salt was shown to be due to the

sodium cation rather than its anion, since different sodium salts yielded similar results. Isotonic saline solution was more effective than the hypertonic solution. The mortality was increased when potassium salts were given.

Moyer, *et al.* (21) studied the therapeutic effects of various solutions on the shock of severe deep burns in a small number of dogs. A combination of transfusions of defibrinated blood and gavage of normal saline solution with added sodium bicarbonate was the only form of treatment that prevented shock without inducing the deadly complications of cerebral and pulmonary edema. Whole blood transfusions given during the first day of the burn have been reported (but not proved) to aid in preventing or treating shock in severely burned patients and also to alleviate or prevent the subsequent anemia that frequently appears about the third day after plasma has been used. Pooled plasma is known to induce hemolytic anemia at times in group A, B and A-B patients.

There is a need for critical, objective studies of the relative effectiveness of different therapies. What are the requirements for colloid, sodium, red blood cells and water? The advantages and the difficulties of the "alternate" or "random" patient approach are apparent. Markley, Rosenthal and their colleagues (22) have recently made an attempt in this direction.

At the present time, replacement therapy is usually undertaken with a combination of whole blood, electrolyte solutions and a colloid containing fluid. Attention has focused on the use of plasma expanders, such as dextran and gelatin because the complication of homologous serum jaundice may follow pooled plasma infusions, and because plasma may be in short supply in time of mass casualties. The widest experience has been with dextran and it has proved generally satisfactory. The bleeding tendency which has been observed when dextran has been infused in moderate amounts into normal volunteers has not been reported in burned patients receiving large amounts of dextran in the first 2 days after injury. Human serum albumin is preferable to the plasma expanders, but is not generally available in adequate quantities.

Many factors must determine the amount and rate of fluid, colloid and electrolyte replacement; the extent, location and depth of burn and the age, weight and prior health of the patient are the most important.

We have found the clinical appearance of the patient, the degree of hemoconcentration and his urinary output useful

therapeutic guides. All patients with extensive burns should be placed on constant bladder drainage immediately after admission and an accurate hour-to-hour record of the urinary output should be kept during the first 24-48 hours. Renal blood flow and urine output may be markedly reduced at a time when the peripheral pulse and blood pressure are normal, and before other definite signs of impending shock are seen. When the urine output is good, one may feel sure that the general circulation is good. If, however, the urine output fails, one must anticipate early failure of the general circulation.

Since the greatest fluid loss occurs in the first few hours, it is during this time that the physician has the best opportunity to prevent shock. Fluid therapy should be started at once in seriously burned patients. It seems that most patients with burns involving up to 20-25% of their body surface can be treated satisfactorily with a sodium chloride, sodium bicarbonate (or citrate) mixture if treatment is started early. Often this can be given orally, in concentrations of 75 mEq. Na and 50 mEq. Cl/L. However, if the patient is in shock or nauseated, oral fluids alone are inadequate since gastrointestinal function may be impaired; intravenous fluids should be started. When the usual peripheral veins are not available there should be no hesitation about cannulating a vein, using a polyethylene cannula. No hard and fast rule for the amounts, rates and types of replacement therapy can be given. As indicated above, there are many factors which influence the loss of fluids, electrolytes, erythrocytes, etc., and the reaction of the patient. Therapy should be individualized. However, experience shows that a general pattern of loss and response may be anticipated, and on this basis a number of formulas has been devised as guides to replacement therapy. One of the most widely used is that of Evans (23), based on the extent of the burn and the body weight of the patient. This formula calls for 1 cc. of colloid solution (such as dextran) per kg. body weight to be given intravenously during the first day, and one half this amount the second day. Equal amounts of isotonic saline solution are given, and, for an adult, 2,000 cc. of 5% glucose in water on each of the first 2 days. (Evans and his colleagues have used sodium chloride solutions while we, Moyer, Abbott, Reiss and others use a mixture of sodium chloride and sodium bicarbonate or lactate.) If the burn is deep and extensive, half the "colloid" is blood. The saline and glucose solutions are given orally or intravenously, depending on the severity

of the burn, the state of the circulation, etc. If oral fluids can be taken, the saline is given mixed equally with the glucose and water. The upper limit of the volume of fluids which can be safely given is that quantity indicated by the formula for a 50% burn. For a 70 kg. man, this would amount to 3,500 cc. of colloid, 3,500 cc. of saline and 2,000 cc. of glucose in water the first day; 1,750 cc. colloid, 1,750 cc. saline and 2,000 cc. water the second day. The fluid replacement should roughly follow a schedule which approximates the rate of fluid loss: one half the first day's requirement is to be given in the first 4-6 hours; one quarter in the next 6 hours and the last quarter in the next 12 hours. *"It is emphasized strongly that the simple formula presented here for calculation of fluid and salt requirements is only a guide to therapy and not to be considered an infallible rule"* (Evans). The rate and type of fluid administration is based on the clinical findings, the hourly urine output and the hemoglobin or hematocrit concentration (measured every 4 hours).

In patients with respiratory injury and/or serious cardiac disease, colloid and salt administration should be sharply limited to amounts which ensure adequate response. The salt solution should be given orally if possible.

If severe shock is already present at the time of beginning therapy, or if it develops during therapy, the fluid must be given rapidly to improve the circulation. Caution must be used, of course, in infants and old people, in patients with respiratory tract injury and in patients with heart disease. In the previously normal adult in shock and with a large burn, as much as 2,000 ml. may be given in an hour without danger of overloading the circulation.

Evans has called attention to acute gastric dilatation as one important complication of shock. It may be manifested by a failure of the patient to respond to what would appear to be adequate fluid therapy. Decompression, lavage of the stomach and replacement of the "lost" water and electrolyte will relieve the situation.

There is still controversy over the relative quantities of blood, plasma expanders, electrolytes, fluids, etc., to be used in the treatment of burn shock. Some would recommend less blood, others more saline, some less dextran or plasma. As stressed earlier, controlled studies in patients are needed. Any "formula" must be used only as a guide to shock therapy; for any given patient, therapy must be individualized.

ADDITIONAL THERAPEUTIC FACTORS

Vitamins

Limited experimental work in animals has indicated that the B-complex vitamins and ascorbic acid may help in burn shock. It has also been found that a great depletion of water-soluble vitamins occurs during the shock of burns as well as in traumatic shock in humans. It appears reasonable to administer large doses of these vitamins during the early period.

Position

The circulation of the brain is improved in shock by raising the foot of the bed even though by this maneuver the blood volume may not be materially altered. It is especially important to do this during the first few minutes while more effective methods of treatment are being started.

Oxygen

There is no evidence that inhalation of 100% oxygen alleviates the tissue anoxia of shock. Oxygen has been widely used in the treatment of burn shock, but the only clear indication for its use is when the burn is complicated by respiratory tract injury.

External Temperature

Overheating or undercooling are both detrimental to a person in shock. The best environmental temperature appears to be about 24 C.

Sedation

It has been noted that the mortality of burned dogs was increased when the usual doses of morphine or barbiturates were given. These findings, and others of a similar nature, indicate that morphine and other sedatives should be used cautiously in patients with impending or actual burn shock. When used, administration by the intravenous route is essential, since the absorption of subcutaneous or intramuscular medication is unpredictable. The action of anesthetics is also

unpredictable; they are dangerous in burn shock. There is almost no occasion to use general anesthesia at the time of the first dressing.

Pressor Drugs

These drugs are at present considered to be of no value in the treatment of burn shock and may be definitely harmful.

Adrenal Cortical Extract

There is no evidence to indicate beneficial effects from adrenal cortical extract, ACTH or cortisone in most patients during the early phase following burns. A patient with adrenal exhaustion might rarely be encountered; he would be benefited by adrenal cortical extract.

RENAL COMPLICATIONS

One of the most serious complications in patients with severe burns is impairment of renal function, evidenced by oliguria, anuria and azotemia. Albumin, hemoglobin and casts are frequently found in the urine of these patients, but large numbers of white or red blood cells are rare. Detailed discussions of the kidney lesions are presented by Goodpastor, *et al.*, Lucke and, most recently, Sevitt (24). The changes in renal function and morphology are similar to those commonly associated with acute tubular necrosis from other reasons. The cause of the lesion in the burned patient appears to be reduced renal blood flow and anoxemia associated with peripheral circulatory failure. Additional factors may be acidosis, methemoglobinemia, methemoglobinuria and the presence of circulating red cell stroma.

Every effort must be made to prevent the occurrence of shock or, if shock is present, to combat it vigorously and immediately. The most important prophylactic treatment of serious renal dysfunction is early adequate correction of blood, fluid and electrolyte deficits. It appears reasonable to inject reducing agents such as ascorbic acid to reduce the methemoglobin to oxyhemoglobin, but this procedure has not been tested.

Patients with serious burns and renal failure present many therapeutic problems arising from (1) the early increase in fluid volume and electrolytes in the burned area, (2) the pres-

ence of contaminated and infected wounds and (3) rapid tissue breakdown with release of nitrogenous metabolites and potassium. Similar problems are faced in patients with other types of extensive injuries and renal failure, such as were seen in large numbers during the Korean War. Among these patients, mortality was high, about 50%. Malnutrition, increased wound infection and delayed healing were observed. The difficulties encountered and therapeutic methods adopted have been described by several investigators (25). These methods include the use of calcium gluconate intravenously for temporary control of rapidly developing hyperkalemia, and extracorporeal dialysis by an artificial kidney for more lasting control of hyperkalemia and uremia. The same principles are adaptable to the burned patient.

Although serious renal failure is uncommon in patients with extensive burns when adequate "antishock" therapy is carried out, renal dysfunction will be a frequent problem under conditions of war or mass civilian disaster since, under these circumstances, treatment of the injured patient often will be delayed.

HYPERPYREXIA IN BURNS

Patients with burns frequently have a period of high fever (over 41 C.) in the first few days after the burn. This is observed in adults only when the burns are extensive, but in children, and particularly infants, it may occur even when the burns are small. Delirium, stupor, convulsions and/or coma should suggest the possibility of hyperpyrexia. The oral temperature may be misleadingly low because of mouth breathing; rectal temperatures should be measured in all such patients. Infants in particular should have hourly rectal temperatures taken during the first 24 hours, 2-hourly temperatures during the next 24 hours, and not less than 4-hourly temperatures for the next week.

The pathogenesis of this early high fever is probably due to the inflammatory reaction in the tissues, but in most instances there are no clear-cut signs of infection, local or systemic. Pyrogens in intravenous fluids are responsible only rarely, shock and dehydration are occasionally involved and, in some instances, the fever may be cerebral in origin. Both increased heat production and a decreased ability of the patient to lose heat are involved.

The burn patient cannot tolerate a high fever for long. Prompt and energetic treatment directed toward cooling the patient should be started whenever the rectal temperature is above 40 C. His dehydration should be corrected, he should be sponged with ice water and fanned. If less than 50% of the skin is exposed, it may be necessary to remove some of the dressings. Another useful method of cooling the patient is to enclose him in special blankets constructed to allow the circulation of cooling liquids through them. Aspirin may be given in fairly large amounts.

GASTROINTESTINAL TRACT ULCERS

The occurrence of ulceration of the intestinal tract following burns has been recognized since Curling's description in 1842. Ulcers of the esophagus, stomach and duodenum have been observed, singly and in combination (26). They appear the first week or two after injury. Under present therapy, it is an uncommon, but serious, complication.

The etiology of the gastrointestinal ulcers has not been determined, although various theories involving nervous, infectious, toxic, hormonal (viz., adrenal activated), circulatory and embolic factors have been suggested. The symptoms may appear rapidly and include epigastric pain, hematemesis and melena. The possibility of these ulcers should be suspected in the seriously burned patient with upper abdominal symptoms. Many of the patients can be treated satisfactorily "medically" but some, either because of massive hematemesis or perforation, will require operative treatment.

DISTURBANCES IN FUNCTION OF THE CENTRAL NERVOUS SYSTEM

Delirium, stupor, coma, convulsions, hyperpyrexia and Cheyne-Stokes respiration—symptoms suggesting central nervous system injury—have been seen in patients with severe burns. There have, however, been few reports of the pathologic changes of the nervous system in such patients.

Walker and Shenkin (27) studied a small group of patients who died of sudden respiratory arrest. All 6 had been disoriented, maniacal or stuporous. Five of the patients died on the fourth day, the sixth on the sixty-second day. Postmortem examinations showed diffuse cerebral edema with evidence of

marked increased intracranial pressure. Walker and Shenkin's patients showed generalized edema as well as cerebral edema, and it is possible that the cerebral changes were a nonspecific effect. Gibson has also noted cerebral edema in patients dying of burns. Walker and Shenkin described marked changes in the smaller blood vessels consisting of degenerative changes of the endothelial lining, with occasional breaks and perivascular petechial extravasations. The ganglion cells throughout the brain showed toxic degeneration, but the most striking changes were in the cortex and hypothalamus, particularly in the latter.

Christophe (15), as mentioned, has reported, in a series of complicated cross-circulation experiments in dogs, histologic changes in the paratuberian centers, especially at the paraventricular and supra-optic regions of the anterior thalamus of the brain of the unburned animal. These he attributed to a "toxin" originating in the burned tissues. These experiments of Christophe have not been repeated by other workers, but Malm and his colleagues found major histologic changes in the hypothalamic nuclei of unburned dogs healthy by standard criteria. These changes were similar to those considered by Christophe as consequent to burning.

EMOTIONAL DISTURBANCES

The severely burned patient may develop serious emotional disturbances engendered by fright, discomfort, pain, fear of disfigurement and fear of death. Hamburg, *et al.* (28) have discussed some of these problems and have pointed out the importance of the prevention and treatment of such problems in the recovery and rehabilitation of severely burned patients. Close personal contact between physician and patient, guidance, reassurance and occupational therapy are important factors. For some patients, psychiatric help will be required.

SURFACE TREATMENT

Primary Local Treatment

The treatment of the burned skin has always been of paramount interest to the burned patient and his physician. The injury is obvious; the associated pain may be intense. Small wonder that the earliest references to burn are solely concerned with the treatment of the locally injured area.

"You must boil the tender roots of the ilex, and if their bark be very thick and green, it must be cut into small parts, and having poured in white wine, boil upon a gentle fire, until it appear to you to be of the proper consistence, so as to be used for a linament. And it may be prepared in water after the same manner. Another, not corrosive:—Old swine's seam is to be rubbed in by itself, and it is to be melted along with squill, the root of which is to be divided and applied with a bandage. Next day it is to be fomented; and having melted old swine's seam and wax, and mixed with them oil, frankincense, and the shavings of lotus and vermilion, this is to be used as a liniment. Having boiled the leaves of the wakerobin in wine and oil, apply a bandage. Another:—When you have smeared the parts with old swine's seam let the roots of asphodel be pounded in wine and triturated, and rubbed in. Another:—Having melted old swine's seam, and mixed with resin and bitumen, and having spread it on a piece of cloth and warmed it at the fire, apply a bandage. When an ulcer has formed on the back from stripes or otherwise, let squill, twice boiled, be pounded and spread upon a linen cloth and bound on the place. Afterward the grease of a goat, and fresh swine's seam, spodium, oil, and frankincense are to be rubbed in."—Hippocrates (29).

The number of agents which have been applied to burns is vast; almost "everything" has been tried. Although there is no universal agreement as to the best primary local treatment, certain fundamental concepts are now widely accepted and have led to a more nearly unified view of treatment. It is clearly recognized that the therapy of the local and general disturbances must be continually integrated from the time of injury to the end of convalescence. Their separation in this discussion is purely artificial and a matter of editorial convenience. One should try to correct the many physiologic and pathologic changes brought about by the burn. External and subcutaneous fluid loss should be limited, further contamination prevented and infection controlled. Any surface application should be rapid and relatively easy to apply and the least possible attention should be required thereafter. The spontaneous epithelization of second degree burns should not be delayed; the separation of slough from deep burns should be hastened; the agent applied should not interfere with early surgical excision. There should be no adverse local or systemic effects. Absorption of many substances from the surface of both second and third degree burns has been demonstrated. Therefore, any agent applied to a burn should either not be absorbed, or, if absorbed, it must be nontoxic. At the present

time, compression dressings or exposure to air seem to best meet these requirements.

Tannic acid (30), tannic acid and silver nitrate, gentian violet and "triple dye" displaced almost all former methods of primary surface treatment in this country during the period 1925-1939. Tannic acid was introduced by Davidson principally to fix postulated (but unproved) toxins in the skin. Another advantage soon noted was that the early external fluid loss of superficial burns was markedly reduced.

The impact of tannic acid was great. A report of the Medical Research Council of Great Britain in 1929 concluded that the tannic acid method for the treatment of burns was one of the most important advances made in modern therapeutics. However, evidence accumulated in the late 1930's and early 1940's that tannic acid was absorbed from the burned areas and produced serious liver damage.

Aldrich had introduced "triple dye" (a mixture of brilliant green and acriviolet, the latter a loose chemical combination of acriflavine and crystal violet) to control infection of the burn wounds.

> "While the new dye (triple dye—Ed.) is as superior to gentian violet as gentian violet is to tannic acid, I do not believe that it is the final answer to all the problems presented by a burn patient. I'm certain, however, that the conception of the burn as an infected surgical lesion is correct and that it is infection rather than absorption of a split protein which causes death in burns. . . ." (31).

But local toxicity of triple dye and tannic acid was suggested in the late 1930's. In addition, wound infection was not well controlled by these agents. At about this time (1937), Allen and Koch introduced compression dressing technic. They stressed that a burn is analogous to other traumatic open lesions (32). Gentle cleaning, application of a nonadherent compression dressing and splinting are the initial steps which they advised for converting the burn from a contaminated open wound into a closed clean wound. They felt that the compression might reduce both external and subcutaneous fluid loss, that infection was reduced by careful initial cleansing and by the rest and protection furnished by the bulky dressing.

Sulfonamides were introduced into the treatment of burns as soon as they became available. Some sprayed a sulfanilamide solution that formed a semitransparent eschar; others used various sulfonamide ointments or powders. Gurd com-

bined the cleaning and compression method of Allen and Koch with a sulfathiazole "cream." Others used preformed films containing sulfonamides. New antibacterial agents, such as propamidine were discovered and used. Finally, the antibiotics were used locally, singly or in various combinations.

The exposure of burns to the air, an old, but forgotten method was reintroduced by Wallace in 1948 (33).

There is no evidence that the amount of external or subcutaneous fluid loss in the first day following burning is lessened by either the compression dressing or exposure technics as presently employed. This is a serious deficiency which is particularly important in the occasional infant with an extensive weeping superficial burn. The following experience illustrates the point:

A 22-month-old child, weighing about 15 kg., was admitted with hot water scalds involving 25% of his body surface. The burned areas were oozing plasma-like fluid at a rapid rate; there was little subcutaneous edema. Plasma therapy was started, after which petrolatum compression dressings were applied. The external loss of fluid was not arrested so that the bandages continued to be soaked with fluid during the first 36 hours. During this time, 2,000 cc. of plasma were administered with no hemodilution (this amount of plasma represented approximately 3 times the plasma volume of the child). Despite this, he was in and out of shock all day. Would that some nontoxic agent could be applied to such burns to stop such an ooze! Perhaps some combination of a physiologic agent to produce rapid clotting of the exudate and another agent to prevent lysis of the clot would be effective. Systemic adrenal hormone or ACTH therapy does not modify the fluid loss.

Opinions differ as to the relative merits of early surface treatment of burns. Some favor exposure to air, while others prefer dressings, but more controlled observations are needed. Both methods have, of course, the same objective—to provide the conditions necessary for quickest and safest healing of the burn. How this is theoretically accomplished by each method is detailed below. Some of the possible advantages or disadvantages of each method will be apparent. The exposure method is preferred by us for burns of the face and perineum; the compression dressing method for burns of other areas, particularly extensive deep burns, circumferential burns of the trunk and burns of the hands or feet. "The thoughtful surgeon

can often employ either alone or both in conjunction to the patient's advantage. The effort, so far as I can determine now, should be focused on the production of a dry wound, free from infection, and early grafting of the full thickness burn"—Evans (34). No fixed routine should be established for all cases. A clear understanding of the underlying principles will make it clear when variation in methods should occur.

On arrival at the hospital, the burned patient should be placed on a sterile sheet, his clothing removed by gowned and masked attendants and his burns covered with sterile sheets. The area and depth of burn should be carefully estimated and recorded. He should be weighed, or a recent weight ascertained.

Possible respiratory damage or carbon monoxide poisoning should be sought. If he is conscious and in pain, but not intoxicated or in shock, or suffering from carbon monoxide exposure, respiratory tract damage, morphine in small doses should be given intravenously slowly. The intravenous route is used because the absorption of medication given subcutaneously at this stage is unpredictable. If the complications mentioned above are present, Demerol®, not morphine, should be used. Appropriate fluid therapy is started. When the patient's general condition is satisfactory, definitive local therapy is begun.

Compression Dressings

Compression dressings must be applied under strict aseptic conditions. The burns should be inspected and cleaned gently with bland soap or detergent with hexachlorophene or other similar agents. This can be done satisfactorily under light sedation or in some cases with no sedation at all. When the patient is extensively burned, or when large numbers of patients must be treated, washing may be omitted to decrease the handling and dressing time.

Fine-mesh gauze, dry or impregnated lightly with a bland ointment, such as petrolatum, should be applied directly to the wound. Sterile absorbent padding should be placed over this, so that even compression can be secured by means of a firm, wide, stretchable bandage. The padding may be obtained in large rolls of material from 1 to 2 cm. thick and 20 cm. wide, or in the form of large pads. The latter are especially useful for dressing the chest and trunk and for dressing all areas when time must be cut to the minimum, e.g., during

military or civilian catastrophes (35). In applying a compression dressing, the tension must be uniform but care must be taken to use no more pressure than is required. The improper application of a "pressure dressing" to a burn of the hand, forearm or arm may result in prolonged stiffness of the fingers, or even to Volkmann's contracture. The limb must never be wound circularly with long gauze bandages. *Soft padding must be applied very thickly and very loosely first. Then this padding may be firmly but gently and uniformly compressed by circular elastic or elastic weave bandages.*

Since the swelling occurs in areas adjacent to the burn as well as in the burn itself, these areas must be incorporated in the dressing. In burns of the extremities, the dressing must extend distally to cover completely the hand and foot, even if these areas are not burned; otherwise, the venous return from the most distal areas will be obstructed, and as the back pressure builds up, the arterial blood supply will be impaired. Elevation of the extremity may help to prevent stasis and discomfort. An external splint should be added over all extremities. When the hands are burned, a suitable hand and forearm splint should be incorporated in the dressing. Care must be taken to avoid uneven compression and "pressure points."

In burns of the chest and abdomen, a "pressure dressing" cannot be applied without interfering with the patient's respiration, so a firm, bulky, occlusive dressing without pressure is used here.

The dressing must be put on carefully and sufficient adhesive tape must be used to hold it firmly in place. Colebrook (36) has shown that the incidence of added infection was 10 times as high in burns which were found to be imperfectly covered when redressed as in those which were well covered during the interval between dressings. Pressure dressings which remain dry on the outside prevent the entrance of additional organisms. Saturation of the dressing has been minimized by the incorporation of a cellophane membrane or a layer of water repellent material.

The original compression dressing should be left in place for five days. When it is removed, the status of the wound is appraised and the further needs of the patient are estimated. Superficial second degree burns will usually be healed in 2 weeks; excision of the full thickness area and skin grafting should be carried out early according to the criteria and

methods mentioned under "Separation of Dead Tissue from Deep Burns," page 30.

Exposure Technic

Exposure of burns to the air is one of the oldest known surface treatments. In recent years, however, its use had been restricted to burns of the face and perineum until the work of Wallace in 1948 (33). Impressed by the healing of exposed face burns, Wallace extended this treatment to burns of other regions. This technic has aroused considerable interest, since it may be the most practicable method for dealing with mass burn casualties in military or civilian catastrophes.

The treatment of burns by exposure to the air is based on the consideration that bacterial growth is inhibited by a dry, cool surface. Rapid drying of the burn surface is therefore necessary. This requires actual *exposure* of the involved area to the air. *Best results have been reported when the air is warm and dry.* Considerable ingenuity is often required to obtain satisfactory exposure which at times may not even be possible, e.g., in circumferential burns of the trunk. The exudate of partial thickness burns usually dries in 24-48 hours when properly exposed and forms hard crust. Full thickness burns with little external leakage dry rapidly without crust formation. Relative immobilization of the involved area is required, not only for the usual reasons in regard to wounds and wound infection, but to avoid cracking the surface. Cracks allow contamination, and spreading infection may ensue.

The exposure method, if properly carried out, is not simple. Wallace has emphasized that nursing care must be vigorous, particularly during the first 48 hours. Aseptic technic should be followed rigidly until the burns are dry.

When a patient is to be treated by the exposure method, the burned areas and surrounding intact skin are gently washed with a bland soap or detergent with sterile water as soon as the patient's general condition permits. This should be done under aseptic conditions. After rinsing with sterile saline, the excess saline is gently sponged. The patient is then placed in a sterile bed in a position which exposes the burned areas as completely as possible, and the burned regions are immobilized. As mentioned, this may require considerable ingenuity. During and following drying, every effort should be made to prevent cracking.

Epithelization of partial thickness burns progresses under the dried crust, which is gradually cast off as the area heals. If cracking occurs and localized infection begins, the crust should be cut away over the involved area and the uncovered part gently sponged. On continued exposure, such an area may dry out. If the infection spreads, the crust should be completely removed and the area treated by compression dressings.

Sloughing of the dried full thickness burns usually begins spontaneously about 2-3 weeks after injury. It may be delayed in deep burns over areas such as the back and buttocks where the edges of the slough become soft, crack, and infection may start. One should not wait for spontaneous sloughing. The same principles of early wound closure apply which were stressed in the preceding section on compression dressings.

SEPARATION OF DEAD TISSUE FROM DEEP BURNS

Deep burns should be covered with skin grafts at the earliest possible time after injury. Until recently, most surgeons have waited for spontaneous sloughing of the burned skin and formation of granulation tissue. This process is too slow and may lead to excessive scars, contractures and deformities. In addition, until epithelization is complete, infection and disturbances in nutrition, red blood cell formation and other important bodily functions persist.

Chemical Debridement

Early separation of the slough has been accomplished occasionally by the application of various proteolytic enzymes or certain acids. Unfortunately, no dependable preparations which will rapidly and safely remove slough are currently available. However, investigation in this field is active.

Surgical Excision

Immediate excision and skin grafting has been carried out occasionally. It is a theoretically sound procedure because after a deep burn is excised and the wound successfully grafted, the area heals in a week or 10 days. However, it has limited applicability because of the further physiologic upset caused by this operation in a patient whose burn may already have caused shock. Further, as mentioned, it is often

impossible to distinguish partial thickness from full thickness burns shortly after injury. Immediate excision, therefore, is indicated only in *obviously deep burns of relatively small area.* Relatively few deep burns come in this category.

A number of surgeons have excised and grafted as early as 2-10 days after injury when the patient's condition has stabilized. The technic has been detailed by Allen (37). In brief, all damaged tissue must be removed down to normal fat, fascia and muscle. Grafting may be done immediately thereafter or delayed for a few days. In patients with extensive burns, these operations should be done in stages. *The results of early excision and grafting have been excellent. At the moment, this is the procedure of choice.*

Severely burned patients may pose difficult anesthetic problems. Cyclopropane is the anesthetic agent we have used most often. Patients with large denuded areas may lose heat excessively during operation; appropriate counting measures should be taken. The use of endotracheal tubes in young children should be avoided wherever possible because of the danger of postoperative edema of the larynx and glottis. Operative procedures should be carried out with the patient on the side, rather than prone, if possible.

HOMOGRAFTS

Homografting, or the use of skin from someone, living or dead, other than the burned individual, has a limited but definite application in the care of the severely burned patient, particularly children. With excised or granulating areas covered, though only temporarily, the general condition of the individual improves so that by the time the grafts dissolve, autografting can be successfully applied. Homografts cannot be made to "take" permanently by the use of ACTH or cortisone. Investigation in the field of homotransplantation is active; if the conditions necessary for the "permanent take" of skin homografts can be established, this would improve our care of the severely burned patient immensely!

POSITIONING

The importance of proper positioning of the patient has been mentioned previously. Elevation of burned extremities, head, neck and upper thorax is useful in lessening edema and

pain. The necessity for proper exposure of burns treated by the exposure technic has been emphasized. If the patient will be in bed for more than a few weeks, steps should be instituted early to prevent foot drop. While on his back, the patient's feet should rest at right angles against a foot board; while prone, his feet should hang over the edge of the mattress to maintain the right angle.

PHYSIOTHERAPY

Intelligent physiotherapy can do much prophylactically and therapeutically in hastening rehabilitation of the burned patient. No attempt will be made here to detail methods; the measures used must be adapted to each patient.

INFECTION

Infection is the major cause of death of patients with deep burns. While the slightly burned skin of superficial second degree burns retains much of the resistance that normal skin has against bacteria, the slough characteristic of deep burns furnishes an excellent medium for bacterial growth. In addition, there may be a marked lowering of the immunologic defense mechanisms of the severely burned individual. An abrupt drop in serum properdin and gamma globulin levels and increased sensitivity to injected endotoxins and bacteria have been observed in burned animals. A severe disturbance in the phagocytic function of the reticuloendothelial system of the burned dog has also been described; previously phagocytized particulate matter is extruded and there is a progressive decrease in efficiency of phagocytosis (38, 39). It seems likely that the "resistance" of the severely burned patient is also lowered, but studies have been inadequate. The burned individual who is also exposed to significant irradiation is particularly sensitive to infection.

At the Royal Infirmary at Glasgow in the early 1940's, Clark and his colleagues found that the hemolytic streptococcus was the predominant infecting organism of burned patients. This corroborated the previous studies of Aldrich in the 1930's at the Johns Hopkins and the Boston City hospitals. However, most studies during the past 15 years at many widely separated civilian hospitals in the United States have shown that the hemolytic Staphylococcus aureus, not the hemolytic strep-

tococcus, is the predominant infecting bacteria. When the hemolytic streptococcus is predominant, it interferes with successful grafting; fortunately, it may usually be eliminated by the use of large doses of penicillin. Actually, burns at all stages are almost always contaminated or infected with several organisms. Aerobic, anaerobic, gram-positive and gram-negative bacilli and cocci may all be found; serial cultures taken from a single burned area show changes from time to time in the variety and proportions of the different organisms. The bacteria most frequently encountered in burn wounds are listed in the table.

VARIETIES OF BACTERIA COMMONLY FOUND IN CULTURES FROM BURNS

Staphylococcus hemolyticus, coagulase positive	Other streptococci
Streptococcus hemolyticus (beta and gamma)	Other enterococci
Bacillus proteus	Pneumococci
Pseudomonas aeruginosa	Bacillus mucosus capsulatus
Bacillus coli	Bacillus welchii
Diphtheroids	Bacillus tetani
Other staphylococci	Other clostridia

Liedberg (40) has divided the infection of the burned patient into 3 subtypes: "(1) colonization, which involves organisms with or without pathogenic potentialities, (2) local invasive infection, which is characterized by local signs of inflammation and (3) general invasive infection, which is characterized by lymphangitis, lymphadenitis, spiking fever, leukocytosis and bacteremia. Suppuration may or may not be a feature of any of these three categories."

Clark and co-workers (41) have emphasized that most infections in patients with burns are "added infections"—that is, a transfer of infective material to the burned areas from sources outside the patient. As prophylactic measures, the following procedures are useful:

1. The masking, gowning and scrubbing of all personnel coming in contact with the patient when his burned areas are uncovered. This procedure is most likely to be violated at the time of admission of patient to the accident floor; rigorous attempts must be made to avoid this.

2. Dressings and operations should be done under strict aseptic technic in a room specifically set aside for the care of burned patients. By these methods, many cross-infections incurred at time of dressing can be eliminated. Ideally, the air coming into this room should be kept at a comfortable temperature; it should be filtered and forced in at a rate such that, theoretically, the entire room air is changed every few minutes. Colebrook and his associates have shown that with such a system it is possible to maintain an almost microbe-free atmosphere in the room.

3. When the compression dressing technic is used, the burns must be *completely* covered at all times. If the dressing becomes wet, contamination from the outside can occur. This can be lessened by incorporation of a nonwettable material in the outer layers of the dressing.

4. Ideally the air on the ward should be routinely purified and its temperature and humidity controlled. This is particularly important when the burns are treated by the exposure technic to allow for the rapid drying of the burned areas.

5. Infected patients should be segregated in separate cubicles.

6. Attendants and visitors with "colds" should be excluded.

7. Immobilization of the burned and adjacent areas should be carried out as described.

8. Maintenance of dry wounds, whether the compression dressing or exposure technic is used.

9. Removal of dead tissue of deep burns and grafting as soon as possible.

10. Maintenance of the nutritive and general condition of the patient. The possible usefulness of gamma globulin injection is under study (39).

11. Prophylactic immunization against tetanus (toxoid or antitoxin as indicated). Although a variety of anaerobic bacteria are commonly cultured from burns, it is the Bacillus tetani among the anaerobic organisms which most frequently leads to clinical infection.

12. Chemotherapeutic agents and antibiotics. As in the handling of patients with other traumatic wounds, certain chemotherapeutic agents and antibiotics are useful adjuvants to, but no substitute for, intelligent surgical treatment of the burned patient. Antibiotics should *not* be administered to *all* burned patients. The use of these substances should be restricted to patients with deep second degree or third degree burns or with respiratory tract injury. Antibiotic prophylaxis and therapy must be combined with early removal of the burn slough and skin grafting.

Penicillin, given parenterally, is the antibiotic usually started on admission. Later, the antibiotics used should be based on bacteriologic cultures and sensitivity tests. Altemeier (42) has noted that the variation in natural susceptibility of different strains of bacteria hinders the selection of suitable antibiotic agents for reasonably

complete antibacterial coverage. Development of resistance must be looked for, as well as overgrowth by species insensitive to the antibiotics being used. The antibiotics are used both locally and systemically. Penicillin is generally effective against the hemolytic streptococcus, but is usually ineffective against most of the gram-negative organisms and the hemolytic Staphylococcus aureus, coagulase positive. Erythromycin and chloramphenicol are most likely to be effective against this latter organism. Among the gram-negative organisms, Bacillus proteus, Bacillus coli and Pseudomonas aeruginosa are most commonly encountered. Infections with the last two may at times be effectively prevented or treated by application of polymyxin locally. A combination of bacitracin, neomycin and polymyxin used locally is currently under evaluation.

SCARS AND CONTRACTURES

Early excision of burn slough and skin grafting, control of infection and intelligent physiotherapy will do much to eliminate contractures and scars; the treatment of established contracture scars is discussed elsewhere (43).

METABOLIC AND NUTRITIONAL PROBLEMS OF BURNED PATIENTS

Malnutrition can be a serious complication of severe injury. When it occurs, surgical procedures and anesthesia become risky, wound infection increases and wound healing and the "take" of skin grafts are delayed. Gastrointestinal and liver function are also usually impaired, convalescence is prolonged and mortality is high. Why should malnutrition occur after injury?

In the healthy adult, the vastly complicated and precisely interconnected metabolic reactions are so finely adjusted, that neither the total bulk of the person, nor any part of him, experiences a measurable net change, chemical or physical, despite their continuous turnover. This situation has become known as the "dynamic steady state." Something happens to disturb these interrelationships after an injury and the "steady state" characteristics become mysteriously modified.

Since Cuthbertson's classic description of the post-injury outpouring of urinary nitrogen and sulfur, a veritable flood of studies have appeared dealing with the biochemical changes following injury (44, 45, 46). Abnormalities have been described in the metabolism of protein, carbohydrate, fat, vita-

mins, water and certain electrolytes. It has become clear that they are closely interrelated and must be thought of as components of a single large process. In general, the biochemical changes are qualitatively similar in patients with a wide variety of injuries. The intensity and duration of these disturbances depend on the severity of the injury and the age, sex and prior health of the patient. Only minor nutritional problems are encountered in healthy patients with superficial second degree burns even when large areas of skin are burned. In contrast, healthy patients with deep burns involving over 10-15% of their body surfaces may show marked nutritional disturbances. It is the seriously injured young man, previously healthy, who shows the greatest metabolic upset; the reaction is less in females, children, the elderly and the malnourished. The reasons for these differences in reactivity have not been determined.

The healthy adult man usually eats food yielding about 2,500-3,000 calories and 60-100 Gm. protein each day. This represents 10-15 Gm. nitrogen. He defecates 1-2 Gm. nitrogen daily in his stools and excretes 9-13 Gm. in his urine; thereby, he is in nitrogen "equilibrium." Following injury, fecal nitrogen excretion is unchanged (unless there is specific gastrointestinal tract damage), but the urinary nitrogen (chiefly urea) increases sharply, reaches a peak at about 5-7 days, and may continue for 3-7 weeks. During this time, the injured patient is in negative nitrogen balance and there is a gradual depletion of his body protein. Thereafter, as he recovers, urinary nitrogen excretion falls and since the patient's intake is often high by this time, he goes into positive nitrogen balance until recovery is complete and his body tissues are restored.

The mechanism of the early increase in urinary nitrogen excretion has been studied in some detail in animals, using tagged amino acids as tracers. These experiments have shown that both tissue build-up and tissue breakdown are going on at increased rates following burning. All tissues do not participate equally in this response; the protein content of tissues with rapid turnover rates changes little, or even, as in the case of the liver, increases. In contrast, the protein content of the carcass, despite its much slower turnover rate, falls and accounts, mathematically, for almost all the extra urinary nitrogen.

The burned patient, in addition to losing excessive amounts of nitrogen in his urine, loses a variable amount of nitrogenous

compounds from the burned area, depending on: (1) depth of burn, (2) extent of burn and (3) infection. Early after burning, there is a large outpouring of protein-rich fluid from weeping, superficial burns, while protein is transiently trapped in the dermal and subcutaneous edema fluid of both superficial and deep burns. If the deeply burned skin is relatively dry, the amount of nitrogenous compounds lost from the skin in the first days is considerably less than that sequestered into and under the skin. Later, during the sloughing and granulating stages of deeper burns, there is a steady loss of nitrogenous compounds from the surface. The loss varies directly with the area of skin involved and the degree of infection. During the period of maximum wound slough and purulent infection, the surface nitrogen discharge may constitute 25-30% of the total, including urinary and fecal excretion.

There are changes in plasma protein concentrations due to shifts and losses of water, electrolytes and plasma proteins soon after burning. Usually, the greatest decrease is in the albumin fraction; if shock is severe, there is often a decrease in prothrombin and fibrinogen concentrations. Later, hypoproteinemia may occur as part of the generalized tissue protein depletion. Again, the albumin fraction is chiefly decreased while the changes in the other protein fractions are variable, depending on such factors as liver function and the presence of infection. Considerable attention is currently being paid to the gamma globulin fraction; an early and often sustained decrease has been noted in the severely burned patient.

It is not only protein metabolism that is altered following injury; increased plasma pyruvic acid concentrations, lactacidemia, hyperglycemia, glycosuria and acidosis may appear early and persist over a period of days. Decreases in muscle and liver glycogen following burns have been described in animals but similar measurements have not been made in patients. It appears that there is a marked increase in peripheral carbohydrate turnover, including an increase in gluconeogenesis. Impaired glucose tolerance and insulin resistance have been reported in some patients with severe burns, particularly if high carbohydrate diets are given. The abnormal carbohydrate metabolism may last days, weeks or months.

Just as there is an increase in protein and carbohydrate turnover after injury, so there is an increased turnover of body fat. Moore and his associates (47) have observed a rapid loss of body fat (presumably due to increased oxidation) during

the early post-injury period. In rats, there is an increased uptake of C^{14} by the perirenal fat of burned animals as compared with that of controls following the intraperitoneal injection of methyl-labeled sodium acetate (48).

In regard to vitamin metabolism, decreases in plasma ascorbic acid concentration, its urinary excretion (*not* followed by a period of increased loss) and "tissue saturation" as measured by intravenous "load tests" have been described. These changes are greatest in the early period following injury, but in some instances continue far into convalescence. Similar changes in urinary excretion and "tissue saturation" have been reported for thiamine and nicotinamide. There is also a decrease in urinary excretion of riboflavin early, which, at times, is followed by a brief period of increased excretion of riboflavin. Later, during the convalescent period when the patient is in positive nitrogen balance, there is also a positive riboflavin balance. No data are available regarding the fat-soluble vitamins.

Abrupt disturbances in water and electrolyte metabolism may be marked following injury. The early migration of fluid into, adjacent to, and from the injured area, have long been recognized. There may be a general increase in measured extracellular space. Serum sodium and chloride concentrations decrease in the early post-injury period; there is a shift of sodium into injured cells with a concomitant outward shift of potassium. Sodium and chloride excretions are initially low, resulting in early positive balances, but later, during the period of resorption of wound edema and diuresis, these balances transiently become negative. The periods of these changes are generally considerably shorter than those of the changes in protein metabolism. Potassium balance is usually negative in the early days after injury but becomes positive gradually as potassium intake is recommenced. Apparent correlation between nitrogen and potassium balances is variable; at times there is more potassium lost early than would be expected from the known potassium:nitrogen ratio in normal tissue. Also, potassium balance may become positive sooner than nitrogen balance. At other times, the potassium and nitrogen balances are more nearly parallel.

We are still not entirely clear as to the mechanisms for the biochemical changes after injury. The urinary nitrogen loss, and thereby the underlying tissue protein depletion, is accentuated by, but is not a direct consequence of, the reduced

food intake which the severely injured patient commonly has in the early post-injury period. There are other factors involved—autolysis of injured tissues, inactivity of the patients, increased oxygen consumption, altered liver function and altered endocrine activity, particularly of the hypothalamic-pituitary-adrenal axis. These matters are discussed in detail elsewhere (49, 50, 51, 52).

Although there are innumerable descriptions of the biochemical changes which occur soon after injury, little objective data are available concerning the clinical effects attributable to these changes. There are conflicting opinions as to the significance of these early metabolic derangements, and attempts may or may not be made to modify the response depending on the viewpoint taken. Since the burned patient must heal his wounds if he is to recover successfully, it would seem that systematic observations of wound healing would provide some objective evidence as to the significance of the early metabolic derangements.

There is little doubt that wound healing is impaired late after injury in severely burned patients who have become overtly malnourished; their granulations are often soggy and friable or, in occasional cases, absent; local exudation and infection are increased; epithelization from the periphery is markedly delayed, skin grafts fail to "take" and donor sites heal slowly. When malnutrition is obvious, other ill effects also occur.

But what of the burn patient who had been previously healthy and well nourished and who suddenly enters a period of profound metabolic alteration? How do his wounds heal in the early period after burning before gross nutritional abnormalities are evident? A few studies in animals bearing on this problem may be cited. Chassin and his associates (53) and Levenson, *et al.* (54) noted a delay in the healing of laparotomy wounds in rats with distant injuries, including severe burns. This was not unexpected—it was over a quarter of a century ago that Carrel (55) demonstrated delayed healing of open wounds in animals with abscesses elsewhere in their bodies. But what of the mechanisms underlying this impairment of healing? Since wound healing involves a complicated intermeshing of biochemical, physiochemical and physiologic reactions, it is likely that the alterations in healing of severe wounds are related to the abrupt generalized metabolic changes characteristically seen following trauma. In

this regard, the changes in protein and ascorbic acid metabolism and adrenal activity seem particularly pertinent.

Chassin and his co-workers found that in young adrenalectomized rats, maintained on a small fixed dosage of aqueous adrenal cortex extract, skin-excision (4 days prior to laparotomy) did not result in depression of the bursting pressure of 5th-day laparotomy wounds, as is the case with nonadrenalectomized rats subjected to the same preoperative stress. This observation is not in accord with the concept that the increased secretory activity of the adrenal cortices during stress tends to maintain homeostasis rather than to cause hypercorticalism.

As mentioned, following severe injury, there is an abrupt and sustained drop in blood and urinary ascorbic acid and an apparent decrease in tissue ascorbic acid saturation as judged by intravenous load tests. Does this represent functional scurvy? If the injured individual is truly physiologically scorbutic, alterations in healing characteristic of ascorbic acid deficiency (hemorrhage, adequate fibroplasia, adequate reticulum formation, but a delay in the maturation of the fibroblasts and production of collagen) should be observed. The guinea pig (as does man) requires an exogenous supply of vitamin C. Accordingly, Levenson and his associates (56) studied the healing of abdominal incisions in control and burned guinea pigs receiving 2 mg. ascorbic acid daily. This is the minimal amount of ascorbic acid necessary for normal growth and normal healing of laparotomy incisions in otherwise normal guinea pigs. The laparotomy wounds in the burned guinea pigs were histologically grossly abnormal—fibroplasia was ample, but hemorrhages were frequent, reticulum and ground substance formation was prolonged and collagen production was scanty and late. These changes were indistinguishable from those in unburned scorbutic animals. Neither reduction of food intake nor administration of large doses of cortisone (10 mg. subcutaneously daily) led to these changes. Finally, administration of large doses of ascorbic acid to the burned animals, 100 mg. daily, beginning a few hours after burning, resulted in normal healing. These results indicate that guinea pigs receiving amounts of vitamin C adequate for normal growth, maintenance and healing, become physiologically scorbutic when severely burned.

The problem of wound infection is closely tied to the problem of wound healing; when wound healing is impaired, wound infection is more common; when wound infection is present,

wound healing is delayed. As described earlier, in the section on Infection (p. 32), there is evidence that severely burned animals have a lowered resistance to infection. The changes in globulin concentrations observed in severely burned patients have already been mentioned; their clinical significance is not yet known.

NUTRITIONAL CARE OF PATIENTS WITH SEVERE BURNS

No attempt will be made here to discuss in detail the nutritional care of patients with burns; rather, only a few general principles will be mentioned. Additional information may be obtained elsewhere (57, 58, 59).

We know in a general way that the requirements of severely burned patients are higher than those of normal persons, and that fairly high dietary intakes should be begun early. If the food intake of the seriously burned patient is low, nitrogen loss is accentuated. However, reduction of food intake alone does not account for all the increased urinary nitrogen loss (60). An increased urinary nitrogen excretion occurs even when there is no reduction in food intake, but the net nitrogen loss is less when the level of intake is maintained than when the dietary intake is low (61).

The food provided must be a complete metabolic mixture, containing adequate amounts of protein, carbohydrate, fat, minerals, water and accessory food substances. But we do not know what the optimal quantitative relationships of the various metabolites should be. It is generally possible to feed most patients orally in a reasonably satisfactory fashion. As mentioned earlier, it is the previously healthy young adult male who presents the greatest nutritional disturbances. No blanket statement can be made as to how soon after injury oral diets should be started; this time would vary, depending on the severity of the burn and the occurrence of circulatory failure. In patients with severe shock, oral feedings would have to be delayed, because of impaired gastrointestinal tract function, for 2-4 days after injury.

Sufficient calories must be supplied to meet the patient's energy expenditure. When insufficient calories are given, the body carbohydrate stores are quickly depleted; then body fat and body protein are burned. Ample dietary carbohydrate and fat should be given, so that the protein can be used for "building" rather than "burning." However, if too high a

carbohydrate intake is given early, serious hyperglycemia, glycosuria and dehydration may occur. This can be alleviated to a large extent if fat is substituted for some of the carbohydrate. It has been our experience that fat should provide from 30-40% of the total calories.

In addition to adequate amounts of fat and carbohydrate, sufficient good quality protein must be eaten. The vitamin requirements of the seriously burned are not known with certainty. We have been giving 5 times the normal requirements of the B vitamins, about 10 times that of vitamin C, and 1 or 2 times the normal requirements of the fat-soluble vitamins. For optimal utilization, each meal the patient eats should be a complete dietary mixture.

Severely burned children can be maintained in excellent nutritional condition when given 1-1½ times their normal requirements. Severely burned women have been kept in good nutritional states by supplying them about 1½ times their normal requirements in regard to total calories and from 2-4 times their normal protein requirements. Young adult males on a dietary intake similar to females will show greater metabolic disturbances for a comparable injury. It would appear that a daily intake of 3-4 times normal protein and about 1½-2 times normal calories may be needed by the very severely burned male (over 20% third degree). This means that a seriously burned young adult male, normally weighing about 70 kg. may need about 275 Gm. protein and about 4,000 calories daily. If renal failure is present, these types of diets (particularly from the point of view of protein, electrolyte and water contents) are not suitable. The problems in the management of such patients have been described by Meroney (62).

The principle of supplying adequate food to patients is simple but the actual carrying out of the principle is often difficult. An "all-out" effort must be made to maintain adequate nutrition. Prophylaxis is preferable to therapy. It is here that the roles of the dietitian and nurses are all-important. They must see to it that the patient actually eats the food that is ordered, for it is not enough that a high-protein, high-caloric, high-vitamin diet has been ordered—it must be eaten. This is not an easy task, since the severely burned patient often has a poor appetite and must be cajoled patiently into eating. However, a surprisingly large amount of food will be taken by such patients, provided attention is paid to his special likes and dislikes and provided an effort is made to serve well-pre-

pared, appetizing meals when the patient is hungry rather than at stated intervals. The control of such diets is exceedingly difficult and we have found it useful to place the major emphasis on high protein, high caloric, high vitamin liquid feedings. These feedings are the main or sole diet. Many reasonably palatable mixtures have been made that contain the food elements known to be necessary for the patient over long periods.

There are several advantages to a liquid feeding regimen. First, a high intake can be provided in a comparatively small volume. If facial burns are present, liquid feedings are much more readily and easily ingested than solid food. The oral liquid feeding regimen has the unusual advantage that it makes the work easier for everyone concerned; it is easier for the patient to take, for the nurse to give, and it is much easier for the dietitian to furnish and record.

If sufficient food cannot be taken orally, either because of lack of appetite, pain or other similar factors, gavage feedings through a narrow plastic inlying gastric tube may be instituted. This is not required often. Once a gastric tube is in place, one is tempted to pour down, at once, large quantities of food. If this is done, marked gastrointestinal upsets will ensue. It is imperative to attain the desired number, concentration and total quantity of gavage feedings by a gradual increase, over several days.

At times, neither oral nor tube feedings are possible in adequate amounts—nausea, vomiting, diarrhea and distention may be limiting factors. Under these conditions, parenteral supplementation is indicated. There are certain fundamental considerations which hold for parenteral as well as for oral feeding—each infusion should be as nutritionally complete as possible and carried out at a rate suitable for best utilization. At present, it is usually impossible to supply enough calories parenterally by the preparations of glucose, fructose and alcohol now available; there is no preparation of fat for intravenous infusion now available for general use, but recent advances make it evident that one soon will be. When the patient is hypoproteinemic and there is a specific indication for raising the plasma protein concentration quickly (e.g., to reduce gastrointestinal tract edema resulting in impaired function), the best way of accomplishing this is by the infusion of plasma or albumin. Albumin should be used in preference to plasma because of certain complications of plasma therapy (homologous serum jaundice, anemia). The plasma proteins can be raised much faster

with albumin infusions than by feeding the patient; the infused albumin participates relatively slowly in the general body protein pool and thereby remains for awhile as protein in the plasma.

The occurrence of anemia early after injury in burned patients with extensive deep burns has already been described. A persistent anemia may also occur in such patients; its chronicity and severity are roughly proportional to the extent of the unhealed full thickness burn. Blood loss from the injured areas (particularly in patients in poor nutritional states whose granulations are friable, edematous and infected) and decreased erythropoiesis are involved in the anemia. If anemia is present, it should be corrected by whole blood or red cell transfusions, but one should not overtransfuse a patient with the expectation of improving the general body nutrition. The nitrogen in infused compatible red cells will enter the body nitrogen pool only when the infused red cells are destroyed—and the average survival of normal cells is about 120 days, not only in normal persons but also in patients in the early "catabolic" and late "anabolic" phases after injury.

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